NARROW BAND UVB AND PUVA THERAPY, A COMPARATIVE STUDY IN PATIENTS WITH CHRONIC PLAQUE TYPE PSORIASIS

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BONAFIDE CERTIFICATE

This is to certify that the dissertation titled " **NARROW BAND UVB AND PUVA THERAPY, A COMPARATIVE STUDY IN PATIENTS WITH CHRONIC PLAQUE TYPE PSORIASIS"** submitted by **Dr. A.MEKALA**, in partial fulfillment for the award of the degree of Doctor of Medicine in Dermatology to the Tamilnadu Dr. M.G.R. Medical University, Chennai, is a bonafide record of the work done by her during the academic period 2006 – 2009.

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DECLARATION

I, **Dr. A. MEKALA** declare that, I carried out this work on, **"NARROW BAND UVB AND PUVA THERAPY, A COMPARATIVE STUDY IN PATIENTS WITH CHRONIC PLAQUE TYPE PSORIASIS"**, at the Department of Dermatology, Govt. Rajaji Hospital, during the period of June 2007- May 2008. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or diploma to any other University or Board, either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for M.D. Degree examination in Dermato Venereo Leprology

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INTRODUCTION

The importance of sun light has been recognized since ancient times. Ultra violet light is a small component of electromagnetic spectrum with narrow band of radiation from 200 – 400nm.

The ultra violet spectrum is further divided into

- Ultra violet C (200-280nm),
- Ultra violet B (280-315 nm) and
- Ultra violet A (315-400 nm).

Artificial UV radiation that allows precise dosing has only been available for the last century.

The phototherapy is a well understood, time proven method for achieving remission in majority of cases. It is a relatively safe method for treatment of :-

- Psoriasis poorly responsible topical treatment
- Rapidly spreading psoriasis,
- Wide spread psoriasis (over 20 % of body surface)
- Severe psoriasis of palms and soles.

REVIEW OF LITERATURE

PSORIASIS

Psoriasis is a chronic, recurrent erythemato squamous disorder involving the skin, nail, joint and mucous membrane. Its a non- contagious disease affecting two percent of the world's population.

Hippocrates provided detailed description of many skin disorders that have dry and scaly eruptions which probably includes psoriasis and leprosy under the name "lopoi" (meaning epidermis).

The two conditions were also grouped together in the old testament with the result then many psoriatics were rejected by the community, the church declared them officially dead and in 1313 Philip, the fair ever ordered them to be burned at the stake. It was not until 19th century that psoriasis was recognized as an entity apart from leprosy.

Robert Willan was first to give an accurate description of psoriasis although Hebra and Kaposi (1876) definitely separated a clinical picture of psoriasis from that of Leprosy.

Galen first introduce the name "psoriasis".

With in the spectrum of cutaneous manifestations, different expression like chronic plaque type, pustular type, guttate type, erythrodermic, inverse type etc., are seen.

The cause of psoriasis is related to the immune system and more specifically T lymphocytes, which is triggered by non specific antigens.

The over active T cells trigger other immune response that cause an increase production of epidermal cells which results in reduction in the cell transit time from basal layer to corneal layer.

The aim of psoriasis treatment is to interrupt the cycle that causes increased epidermal turn over, there by reducing the inflammation & plaque formation.

Psoriasis treatment can be divided into 3 main types.

- Topical therapy
- Phototherapy
- Systemic therapy

Topical therapy is mainly used to treat the psoriasis involving limited area. For larger area of involvement, phototherapy is the preferred treatment, as the systemic therapy like methotrexate, retinoids, cyclosporine etc., are not without much side effects.

PHOTOTHERAPY

As the name suggests, phototherapy uses natural or artificial light. The simplest and easiest form of phototherapy involves, exposing the skin to controlled mount of natural sunlight.

HISTORICAL ASPECTS

- The first report of use of phototherapy in the treatment of skin disorders dates from 1400 BC from India when patients with vitiligo were given certain plant extracts (whose active ingredients include psoralens) and then exposed to sunlight.
- Ancient Egyptians also recognized the beneficial effect of sunlight.
- In 1672, Newton discovered the spectrum of visible light.
- UV light was first discovered in early 1700s.
- In 1890, Modern phototherapy began when Niel Finsen, the father of modern ultraviolet therapy, used a carbon arc source to treat Lupus vulgaris. He was awarded the Nobel Prize in 1903.
- In 1923, Goeckerman, at Mayo clinic, introduced his regimen (artificial broad band UVB + coal tar.)

- In 1947, Fahmy, an Egyptian pharmacologist, isolated psoralen compounds from ammi majus.
- In 1953 Ingram introduced his regimen combining artificial broad band UVB, coal tar and dithranol.
- In landmark of development, in 1974 Parrish et al reported the useful role of high intensity UVA using newly developed Henselar high intensity artificial UVA light in combination with oral psoralen in the treatment of psoriasis leading to advent of PUVA therapy.
- In 1978, Wiskemann introduced broad band UVB tubes for the treatment of psoriasis and uremic pruritus.[1] Broad band UVB was as effective as PUVA in the treatment of psoriasis. Potential carcinogenic effect of broad band UVB made it less popular.
- The break through came after 1988, when narrow band UVB phototherapy was introduced for the treatment of psoriasis by van Weelden et al and Green et al [2,3].

NARROW BAND UVB PHOTOTHERAPY

UVB rays constitute 5 – 10 % of ultra violet light reaching earth. The range from 280 -320 nm.

THERAPEUTIC SPECTRUM

Phillips TL-01 Fluorescent Lamps emits narrow band UVB in the range of 310 – 315 nm, with peak at 312 nm.

* It has a relatively narrow spectrum of emission compared to broad band UVB.
* Has a reduction in the Erythmogenic wave length (290 – 305 nm range),
* 5 – 6 times increased emission of longer UVB wave length, there by resulting in a higher phototherapy index for psoriasis.

PRINCIPLE AND MECHANISM

The mechanism of action[4] of narrow band UVB phototherapy has not been completely understood.

- The major molecular target for UVB is nuclear DNA, with absorption by neucleotides leading to induction of various DNA photoproducts, notably pyramidine dimers.
- the inhibitory action on DNA synthesis.[5]
- Induction of T-Lymphocyte apoptosis –therapeutic effect in psoriasis, eczema, CTCL etc.[6]

- **Other mechanisms:**

- Effects on cell cycle[7]
- Antimicrobial effect and alteration of skin flora.[8]
- Induction of anti inflammatory and immunosuppressive cytokines[9,10,11] - production of IL -10, reduced natural killer cell activity and lympho proliferation.
- It also induces isomerisation of urocanic acid from “trans” to “cis” form, which may be important in the immuno modulatory effects.[12]
- Thus narrow band UVB has therapeutic action in different diseases states involves a combination of effects including changes in cell cycle kinetics, alteration in cytokine expression and immunomodulation.

SOURCE OF NB - UVB

The source of NB-UVB is whole body phototherapy unit is with

- Philips TL-01 fluorescent bulbs (100W)
- Sylvania UV-21

It delivers UVB in a range of 310 – 315 nm with a peak at 312 nm. Its in a reduction in erythmogenic wavelength in the 290 – 305nm.

NB-UVB cabins available commercially either incorporate TL-01 alone or in combination with UVA tubes. Combination chambers takes a longer time to administer a treatment dose.

. Recently shorter tubes of NB-UVB is also available in small area treatment equipments (hand and foot unit, NB-UVB comb) for the therapy of localised body area.

Targeted UVB phototherapy is delivered via a portable unit with UV exposure limited to the affected area like psoriatic plaque or vitiliginous macules.

DOSING SCHEDULE

NB-UVB therapy schedule can be tailored according to the patients skin type and local experience.

There are two regimens that are commonly used to determine the initial dose.

1. Involves determination of individuals minimum erythema dose (MED) by means of separate band of TL-01 tubes.
   - MED is determined[13] by standard method.
   - A template with 20 apertures (10 on each side) of 1.5 X 1.5 cm^2 is made over the back of a cotton suit used by operation theatre staff. The cotton flaps made over the apertures enable to either shut or keep the apertures open by using Velcro. The

source of NB-UVB is whole body phototherapy unit with 24 Philips TL-01 bulbs.

- To determine MED a single panel in whole body unit with 6 bulbs is used.
- All apertures are kept open and back – irradiated with 50mJ of NB-UVB. One aperture is closed and remaining aperture are closed one after the other after delivering 50mJ more than the previous aperture. The dosing schedule of NB-UVB (in mJ) is 50, 100, 150,200,250,300,350,400,450, 500.
- The reading were taken 24 hours after exposure. For 311 nm therapy, the initial dose should be 70% MED.
- MED should be determined before NB-UVB for all conditions except vitiligo
- Patients are treated 3-5 times a week., Although more sittings will be beneficial, 2-3 sittings are more cost effective and hence more acceptable.
- If initial dose is tolerated 40,20, or 10% incremental increase of previous dose is used. When a previous treatment results in erythema, no treatment is given in next schedule. Increment every second and third sittings is effective possibly because sub-erythemogenic dose of UVB is also effective as erythemogenic dose.

2. Another approach, involves a standard starting dose (280mJ / cm^2) with stepwise increase (usually 20%) depending upon the patients erythema response.

- In case of mild erythema, the irradiation dose is held constant for subsequent treatment are until resolution of symptoms.
- The goal of the therapy is to achieve persistent asymptomatic erythema. In case of painful erythema with or without edema / blistering, further treatment is withheld till the symptoms subside. After resolution of over dose symptoms, the dose administered is 50% of the last dose and subsequent increments should be by 10%.

MAXIMUM DOSE [14]

In responsive patients NB-UVB can be given for a maximum of 24 months. After one year, a resting period of 3 months is recommended to minimise the annual cumulative dose of UVB. In children, the maximum duration allowed is 12 months.

INDICATIONS

- Psoriasis
- Generalized vitiligo
- Atopic dermatitis

- Lichen planus
- Chronic urticaria[15]
- Seborrhoeic dermatitis
- Prophylactic low dose NB-UVB is beneficial for photosensitive dermatoses like polymorphic light eruption, actinic prurigo, hydroa vacciniforme, cutaneous porphyrias by providing “hardening photo protective” effect. A typical course involves 10-15 treatment given in early spring.[15]
- Beneficial role has been observed with airborne contact dermatitis[16,17,18] to parthenium hysterophorus, a frustrating problem for both the patient and the physician.

LONG TERM USE AND ADVERSE EFFECTS

Acute:

- Immediate sun burn
- Erythema [19]
- Lesional blistering of Psoriatic plaques. [20,21]
- Pruritus [22]
- Corneal burn[23] if eyes are unprotected
- Reactivation of Herpes simplex virus [24]
- Photoallergic dermatitis

Chronic

- Freckling of the skin
- Photo degenerative changes-reduced dermal hydroxyproline levels and induction of gelatinases and elastin cross-links have been shown.
- Possible increase of skin cancer.- It has been calculated that the long term risk of carcinogenesis with its use may be less than that of PUVA therapy[25].

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COMBINATION THERAPY

NB-UVB Phototherapy may be combined with topical and systemic agents to achieve higher clearance rates, longer disease free intervals and lower carcinogenic risk.

- UVB with Calcipotriol – increases the therapeutic efficacy of phototherapy and reduces the irritation caused by calcipotriol.
- UVB with topical tazarotene gel – promotes more effective, faster clearing of psoriasis when compared to either type of phototherapy alone. Pre-treatment with tazarotene gel for 2 weeks before phototherapy significantly reduces the mean minimal erythema dose for UVB.
- Salt water bath (Balmotherapy)
- UVB with glucocorticoids

- UVB with Systemic retinoids - Combining retinoids with UVB theoretically reduces the risk of carcinogenic effect of UVB.
- Topical emollient application – alters the optical properties of psoriatic lesions, improve transmission of UVB and lead to increase efficacy.
- Combination regimens of UVB therapy with methotrexate or cyclosporine A are not advisable, because both substances increases the possibility of UV induced skin tumors.

PUVA therapy

Psoralens:

Psoralens are clinical compounds derived from certain plants such as ammi majus found in Egypt and Indian plants babachee which is also called as psoralea corylifolia.

Psoralens has been found in more than 30 plants such as Lime, Lemon, Bergamot, parsley, celery, fig, cloves[26] etc.

The most widely used psoralen which is tricyclic furocoumarin is 8-methoxypsoralen (8-MOP, methoxypsoralen, xanthotoxin) – principally of plant origin.

Synthetic form of Psoralen :

- 4,5,8 –Trimethyl Psoralen (TMP,trioxsalen)
- 5 Methoxy psoralen (bergapten, 3-carbethoxypsoralen and angelicin)

Pharmacology :

When taken by mouth methoxsalen (8-MOP) is absorbed by gastro intestinal tract. Increased photo sensitivity is observed one hour after the dose, reaches a peak at about 2 hours and disappears after about 8 hours.[27]

The absorption of 8-MOP is increased by concomitant food ingestion as well as by differences in drug formulation.

It is metabolised in liver by hydroxylation and glucuronide formation and over 90% excreted in urine within 12 hours.[28]

Methoxsalen has a serum half life of approximately 1 hour and rapidly eliminated which prevents photosensitivity[29]. When applied locally 8-MOP rapidly penetrates the skin and can be detected in the urine after 4 hours.[30]

Psoralen Photochemistry : [31]

The major photochemical reaction contributing to the cellular damage, is the formation of monofunctional and bifunctional adducts of psoralen with pyrimidine bases in the DNA. This is mediated by intercalation of psoralen with DNA base pair.

Mechanism of Action :

- The formation of mono / bifunctional adducts results in immediate inhibition of DNA synthesis and cell proliferation.
- Immunological alteration by affecting specific cells like lymphocytes or polymorpho nuclear leucocytes.
- Decrease in the percentage of T – lymphocytes[31,32] following PUVA therapy has been reported :
- Selective cytotoxicity through production of free radicals.
- Stimulation of melanogenesis.[31,33]

Sources of UVA Radiation :

The commonly used sources of UVA are,

- Fluorescent PUVA
- High pressure metal halide lamps

Fluorescent PUVA lamp has emission peak at 352nm although the spectrum of anti-psoriatic activity and phototoxic erythema peaks at 335nm, long wavelength has proved equally effective for clearing psoriasis

Indications

Diseases treatable by PUVA therapy are,

- Alopecia areata [37]
- Atopic dermatitis [40]
- Cutaneous T-cell lymphoma
- Dyshidrotic eczema
- Eosinophilic folliculitis and other pruritic eruptions of HIV infections.
- Graft Vs Host Disease
- Generalised Granuloma annulare[34,35]
- Generalised Lichen planus.[35,36]
- Localised scleroderma
- Palmoplantar pustulosis
- Parapsoriasis

- Pityriasis lichenoides
- Photodermatoses
- Polymorphic light eruption
- Pityriasis rubra pilaris
- Psoriasis
- Seborrhoeic dermatitis[39]
- Urticaria pigmentosa
- Vitiligo

Methods of Treatment :

8-methoxypsoralen in the dose of 0.6 to 0.8 mg / kg body weight is administered orally followed by whole body irradiation after 1 to 3 hours. The initial dose of UVA is predetermined by either skin typing or by determining minimal phototoxic dose. Initiation of PUVA therapy according to the skin photo type,[41]

Skin Phototype		**Recommended Dose(J/cm^2)**
I.	Always burn, never tan	0.5
II.	Always burn, sometimes tan	1.0
III.	Sometimes burn, always tan.	1.5
IV.	Never burn, always tan	2.0
V.	Moderately pigmented	2.5
VI.	Black	3.0

Type I to IV determined by history, Type V & VI are determined by physical examination.

Minimal Phototoxic Dose [42,43,44,45]

MPD is defined as minimal dose of UVA that produces a barely perceptible but well defined erythema when template areas of skin are exposed to increasing dose of UVA ranging from 0.5 to 5 J/cm^2

After 1 to 2 hours of administration of oral psoralen, a template is placed on the patient's buttock as in the area of greatest UV sensitivity. Template consists of 6 to 8 areas of at least 1 cm^2 which can be exposed to increasing dose of UVA. Erythema reading is performed 72 hours after testing, at which psoralen phototoxicity reaction usually reaches its peak. A fraction of its dose, example 50 – 80% is frequently used as a starting dose in PUVA therapy although MPD is more time consuming than phototyping, it allows for more accurate and higher UVA doses during initial treatment.

Protocol for PUVA Therapy :

Various protocols of oral PUVA therapy have evolved as follows,

United States Protocol :

1. First treatment exposure dose is based on skin typing.
2. patients are treated twice or thrice a week.

3. Dose increment – 0.5 to 1.5 J / cm^2 depending on the therapeutic response.

European Protocol :

1. First Treatment is administered after determination of individual's MPD and the initial UVA dose is the patients MPD.
2. Four Treatments are given / week.
3. Two treatments are given consecutive days followed by a rest on day 3 after which treatment is resumed for 2 days.
4. Dose increments are individualised.

Manipal Protocol :

1. The usual starting dose is 4 to 6 J / cm^2
2. Dose increments of 0.5 J /cm^2 are done each time till a maximum of 18 J /cm^2 is reached.
3. Treatment is given thrice or 4 times in a week.

Contraindications for PUVA therapy :

Absolute Contraindication :

1. Pregnancy or in patients trying to conceive
2. Xeroderma pigmentosum.
3. Lupus erythematosus
4. Severe hepatic or renal failure

Relative Contraindication :

1. Personal or family history of melanoma
2. Immuno suppressed patients.
3. Previous exposure to carcinogen such as X-Rays, arsenic etc.,
4. Outdoor workers in relatively sunny areas
5. Children and Young adults
6. Minimal diseases
7. Light skin and fair or red hair
8. Received over 200 PUVA treatment

Protective Measures with PUVA Therapy

During Treatment :

1. Patient should wear specially designed goggles to protect their eyes from UVA radiation.
2. Male genitals[53] must be covered throughout the process.

After Treatment :

1. Patient should wear UVA absorbing wrap-around sun glasses for atleast 12 hours after treatment.

2. Patient should avoid sun exposure to day light for atleast 8 hours after taking the drug. Otherwise should wear heavy opaque clothing or apply sunblock with SPF of more than 15

Acute Side effects

Side effects oral psoralen :

1. Nausea and vomiting
2. Hepatic[54,55] / Renal[56] Effects : no laboratory or HPE evidence of hepatotoxicity / renal toxicity after several years of follow up

Acute Side effects of PUVA :

1. Increased delayed erythema reaction to severe burns with blistering.
2. persistent pruritus.
3. Stinging pain
4. Polymorphous light eruption like rashes
5. Acne like eruption
6. Subungual haemorrhage
7. Hypertrichosis
8. Exacerbation of SLE
9. Exacerbation of bullous pemphigoid

Potential long term risk of PUVA :

1. **Chronic Actinic damage**
    - Photoageing
    - Actinic Keratoses
    - Generalised PUVA lentiginosis

2. **Carcinogenesis**[66]

- Its the major concern about long term and repeated PUVA treatment associated with high UVA dose.
- Due to DNA damage, there is down regulation of immune response.
- In PUVA patients, risk of squamous cell carcinoma but not the basal cell carcinoma is significantly increased and is dose dependent.
- Only a few anecdotal cases of malignant melanoma has been observed in long term PUVA treated psoriatics and no increased risk of melanoma was found in all large-scale studies
- Risk was greater in patients exposed to higher doses of PUVA and in patients exposed previously to other carcinogens like ionizing radiation, UVB therapy, methotrexate, tar, arsenic and appeared to be increasing with passage of time.

- Risk of both melanoma and squamous cell carcinoma in PUVA should weighed against substantial efficacy of PUVA and risk of other therapies.

3. Ophthalmic Effects [57]

- Despite data from animal models, which indicate a risk of premature cataract formation, clinical evaluation shows no increase in lens opacities, even in patients who neglected careful eye protection.
- Majority of prospective studies did not report an increased incidence of lens opacities in patients who used eye protection following psoralen ingestion

Other forms of PUVA Therapy :

1. Solar Irradiation :

In this method 8 MOP is given orally in conjugation with sun light exposure (PUVASOL). A major disadvantage in PUVASOL therapy is difficulty in quantifying UV light.

2. Topical Treatment :

Topical application of psoralen followed by UVA irradiation is given. 0.01% TMP[46] is given 10 – 20 minutes prior to UVA exposure is convenient for topical application[47] because of its weak penetrability.

Advantage of this therapy systemic side effects[49] of psoralen is avoided but limitation are :

1. Application to each lesion is laborious and time consuming.
2. Formation of erythema and blistering is more common with topical psoralen application.
3. Intense irregular pigmentation may been seen at the site of treated plaque.

3. Bath PUVA :

Bath solution are prepared by diluting 50ml of 8-MOP in 100L of bath water.[50]

Patients soak for 15 minutes in this solution and then quickly wipe dry. Immediately patients are given whole body irradiation with UVA.

8-MOP required smaller amount of UVA radiation and yielded fewer side effects.[51]

4. Bath suit PUVA [52]

Advantage of Bath suit delivery of 8- MOP are

- Required only 2L of water and 0.8 ml of 8-MOP solution.
- This treatment can be carried out at home with sun light as UVA source (PUVASOL)

- Irregular pigmentation seen with topical PUVA has been overcome.

Combination Therapy

PUVA therapy can be combined with

1. Acitretin
2. Methotrexate
3. Tazarotene Gel
4. In combination with UVB

Combination therapy helps in lowering the dose of radiation and minimizing the side effects. In addition, retinoids help protect against skin cancer, while methotrexate may increase the risk. Calcipotriol should not be combined since UVA radiation degrades vitamin D3.

AIM OF THE STUDY

This study is conducted in patients with chronic plaque type psoriasis,

- To evaluate the efficacy, side effects and cumulative dose of Narrow Band UVB Phototherapy.

- To evaluate the efficacy, side effects and cumulative dose of PUVA phototherapy.

MATERIALS AND METHODS

The material for this study was from the patients with chronic plaque type psoriasis, attending the skin OPD, Government Rajaji Hospital, Madurai Medical College, Madurai, during June 2007 – May 2008.

The diagnosis of Psoriasis was made on clinical grounds

INCLUSION CRITERIA:

Patients with chronic plaque type psoriasis involving greater than 20% of the body surface area.

EXCLUSION CRITERIA:

For NB-UVB Group:

- Photosensitive disorders
- Previous History or family history of malignant melanoma
- History of skin cancers or photodamage
- History of exposure to inorganic arsenic or ionising radiation

For PUVA Group:

- Age less than 18 years
- Pregnant and lactating women
- Photosensitive disorders
- Previous History or family history of malignant melanoma
- History of skin cancers or photodamage
- History of exposure to inorganic arsenic or ionising radiation
- Significant hepatic, renal or cardiac dysfunction
- Chronic alcoholics
- Active infections like tuberculosis
- Cataract

All patients were explained about the disease and the benefits and side effects of therapy were discussed with them.

Consent was obtained from all patients before initiation of therapy.

All patients were evaluated as follows

1. History
2. General Examination
3. Systemic examination
4. Dermatological examination
5. Investigations
    a. Complete haemogram
    b. Urine analysis
    c. Blood sugar, urea, serum creatinine, calcium and uric acid
    d. Liver function tests
    e. Blood VDRL
    f. ELISA for HIV
    g. Ophthalmic evaluation
    h. X – ray chest

TREATMENT PROTOCOL AND METHODOLOGY :

The study population was divided into 2 groups-Group A and B.

Group A : Narrow Band UVB phototherapy

Group B : PUVA therapy.

PROTOCOLS:

Group A: **(NB – UVB)**

- 20 patients were included in this study.
- All patients were asked to wear UV goggles when inside the phototherapy unit.
- Men were advised to wear genital protection.
- Patients were asked to apply mineral oil on the plaques of psoriasis prior to the exposure of UV light.
- Irradiance of UVB light were recorded on a once a month basis using the standard method of manufacturers of phototherapy unit.
- Initial UVB dose(mJ/cm^2) were calculated according to the skin phototype of the patient.
- The manual method for calculation of time (seconds) to set UVB control panel to deliver the dose is by the following equation,

Time (Seconds) = Dose (mJ/cm^2) / irradiance (mw/cm^2)

- Patients were advised to stand in the centre of the phototherapy unit with their arms rest.
- Patients are instructed to come out of the chamber when the light go out or if they became uncomfortable during the treatment either from burning or stinging sensation of the skin.
- Subsequent dose increments were made at every third sitting at an increment 50 mJ/cm^2 of the previous dose.
- Treatment were given thrice weekly.
- Every patient were monitored regularly after six to eight sittings.

GROUP B (PUVA)

- 20 patients were included in this group.
- Oral 8-methoxy psoralen crystalline tablets at a dose of 0.6mg/kg rounded up to the nearest of 20mg were given.
- After 2 hrs of oral 8-MOP, they were exposed to UVA radiation in the UVA cabinet.
- The starting UVA dose were determined by skin type. Treatment was given twice weekly.
- Patients were asked to wear UV blocking sunglasses during exposure in the cabinet and also for 24 hrs after treatment.
- Men wore genital protection inside the cabinet.

- UVA irradiance of the cabinet was measured monthly using UVA radiometer.
- Exposure time in seconds was calculated using the formula

$$\text{Time in (sec)} = \frac{\text{Required dosage (Joules/cm}^2\text{)}}{\text{Irradiance (mw/cm}^2\text{)}} \times 1000$$

- In our study, all patients belonged to skin phototype IV & V and were started with 3 J/cm^2.
- Subsequent increments of 0.5/cm^2 were given based on response to therapy and presence or absence of erythema.

Follow up

- Patients were followed up every week until cessation of treatment.
- Complete haemogram was done every 2 weeks for the first month and therefore once a month.
- Liver function tests, blood urea and serum creatinine were done every month.
- Adverse effects reported by the patients or noticed were recorded.

Efficacy Assessment

Severity and extent of psoriasis were evaluated using "Psoriasis Area and Severity Index" (PASI).

Severity of Erythema (E), Desquamation (D) and Induration (I) was recorded on a 5 point scale as follows:

0	-	Nil
1	-	Mild
2	-	Moderate
3	-	Severe
4	-	Very Severe

The area of involvement was recorded on a 7 point scale as follows:

0	-	Nil
1	-	< 10 %
2	-	10 % - 29 %
3	-	30 % - 49 %
4	-	50 % - 69 %
5	-	70 % - 89 %
6	-	90 % - 100 %

PASI was calculated as follows

$$PASI = 0.1 (E_H + I_H + D_H) A_H + 0.2 (E_U + I_U + D_U)A_U + 0.3 (E_T + I_T + D_T) A_T + 0.4 (E_L + I_L + D_L)A_L$$

A	-	Area
H	-	Head
U	-	Upper Limb
T	-	Trunk
L	-	Lower Limb

OBSERVATION AND RESULTS

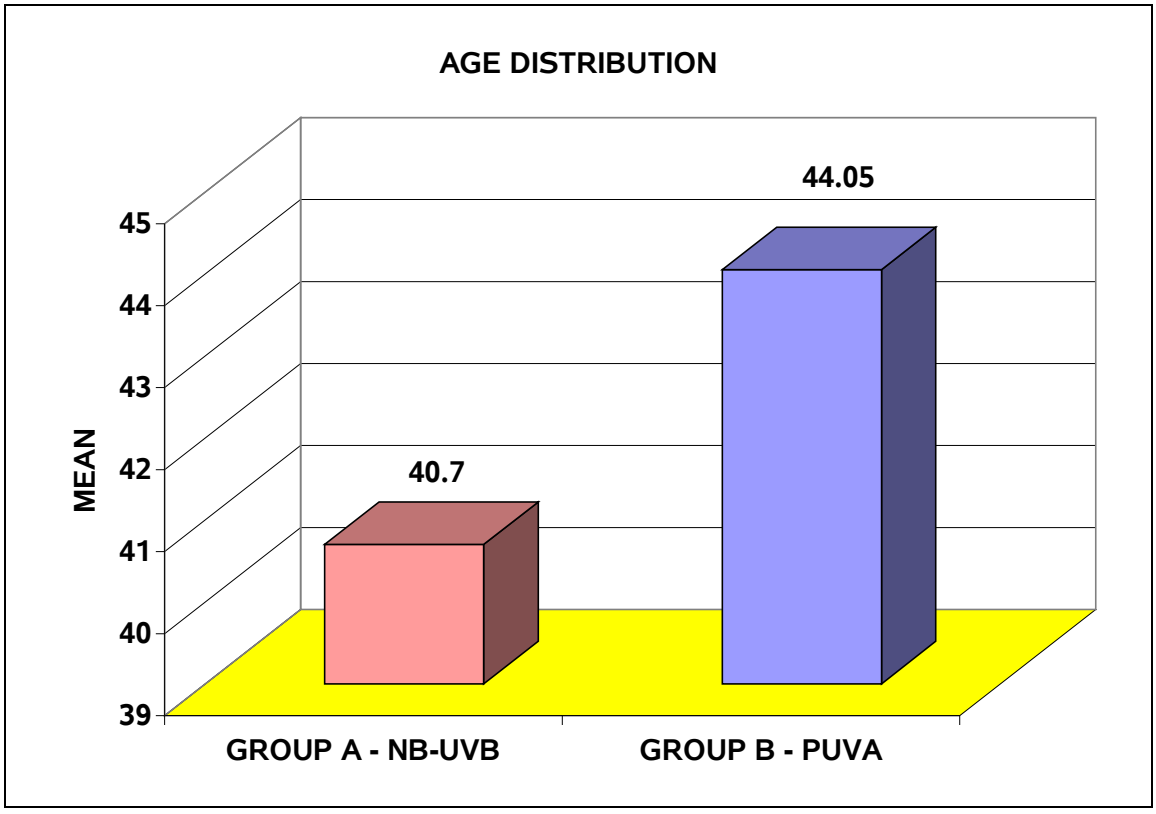
1). Age Distribution:

The range of age was from

- 12 – 70 yrs for group A and
- 31 to 60 yrs in group B

Mean age in our study was

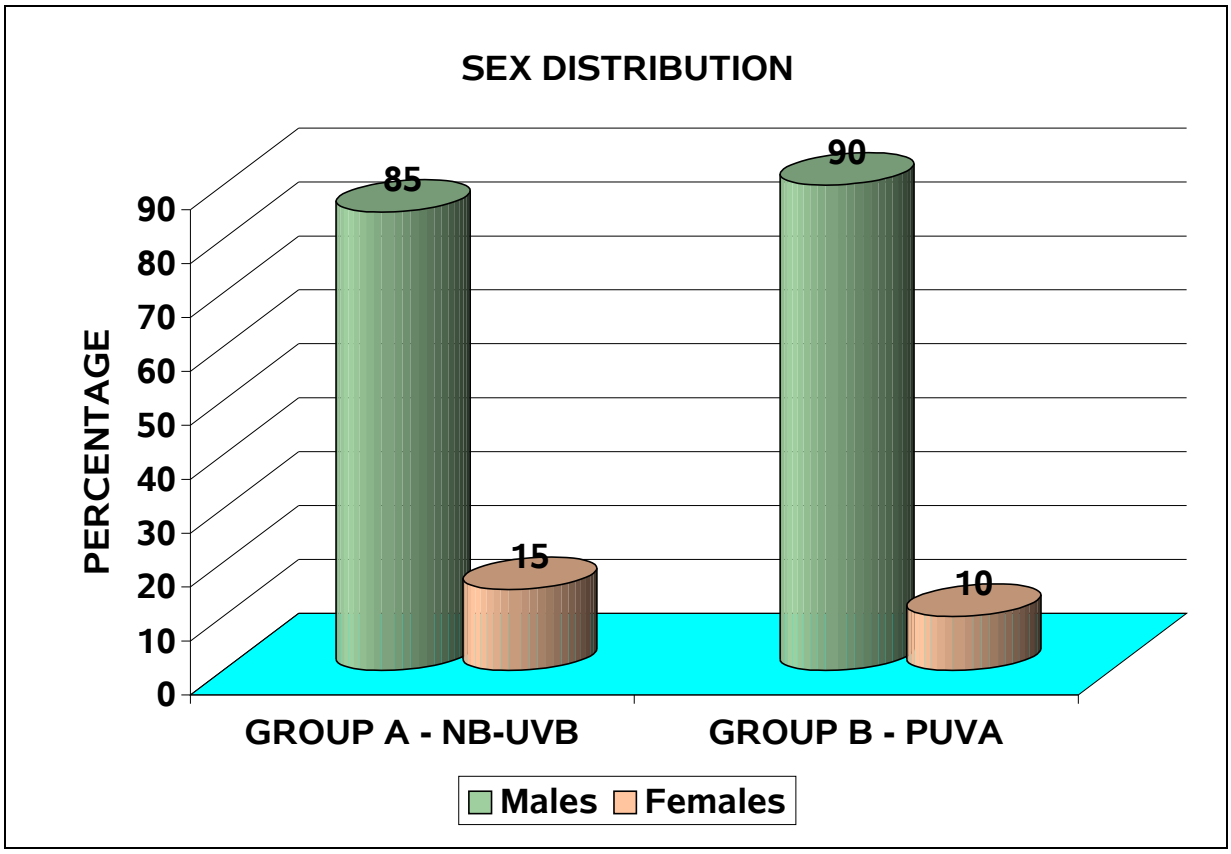
Group A (NB-UVB): 40.7yrs, Group B(PUVA) : 44.05 yrs



2) **Sex Distribution :**

Males out numbered females in both groups.

Sex	GROUP A (NB-UVB)		GROUP B (PUVA)	
	No.	%	No.	%
Males	17	85	18	90
Females	3	15	2	10



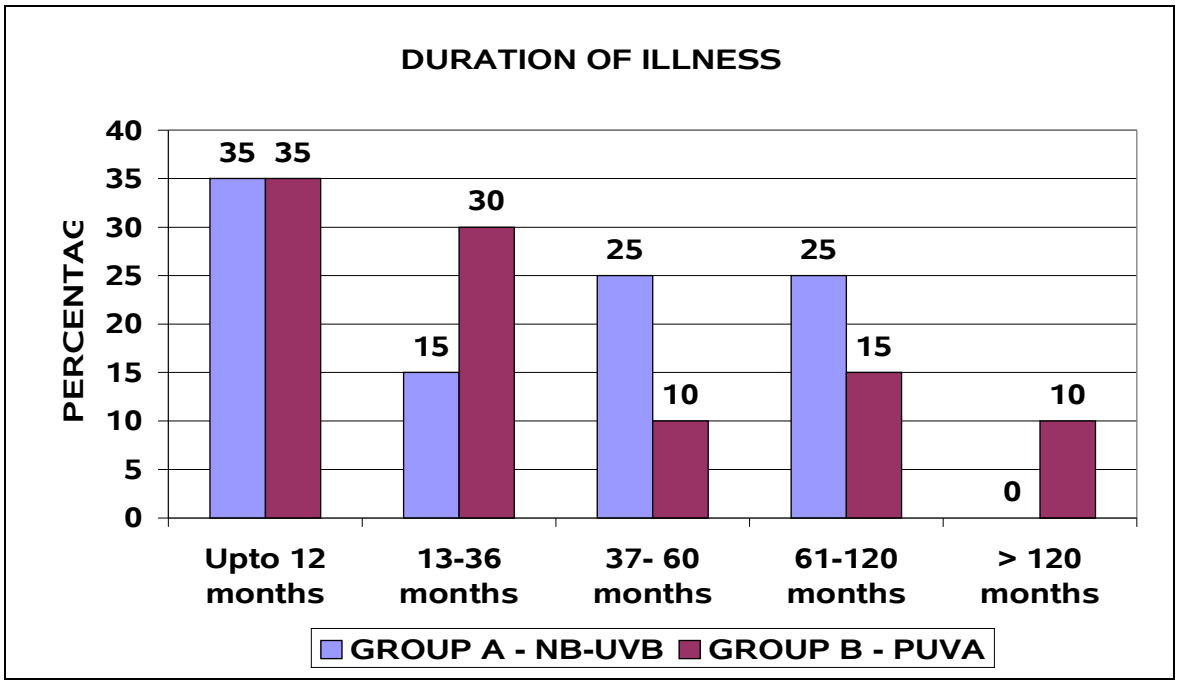
3) **Duration of Illness :**

The range of duration of illness was from 3 months to 15 years and mean was as follows,

Group A : 3.9 (UVB)

Group B : 3.92 (PUVA)

Duration of illness (in months)	GROUP A (NB-UVB)		GROUP B (PUVA)	
	No.	%	No.	%
Upto 12 months	7	35	7	35
13-36 months	3	15	6	30
37- 60 months	5	25	2	10
61-120 months	5	25	3	15
> 120 months	-	-	2	10



4) <u>Family History :</u>

Family history was not present in any of the patients in our study.

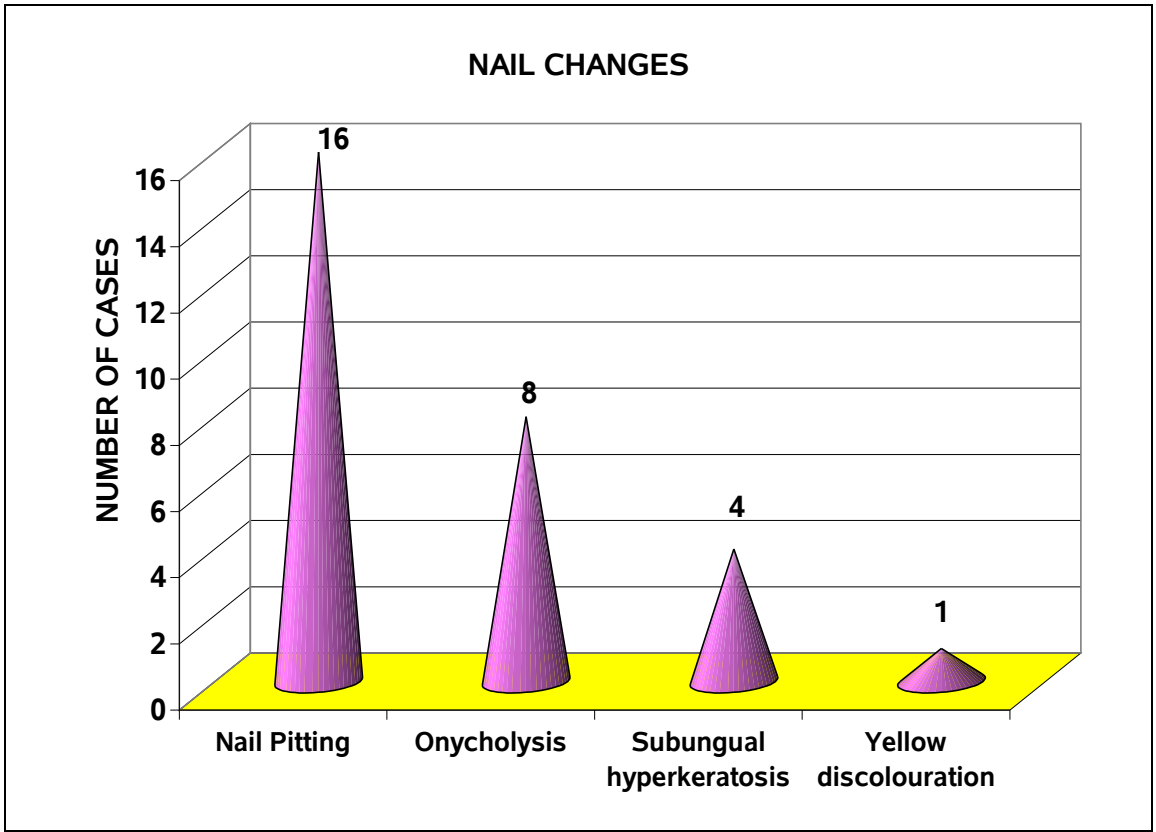
Family History	Group A (NB-UVB)		Group B (PUVA)	
	No.	%	No.	%
Yes	-	-	-	-
No	20	100	20	100

5) Joint Involvement:

Five patients in our study had joint involvement.

- 3 patients had symmetrical poly arthritis,
- 2 patients had peripheral asymmetrical oligo arthritis.

6) Nail Changes :

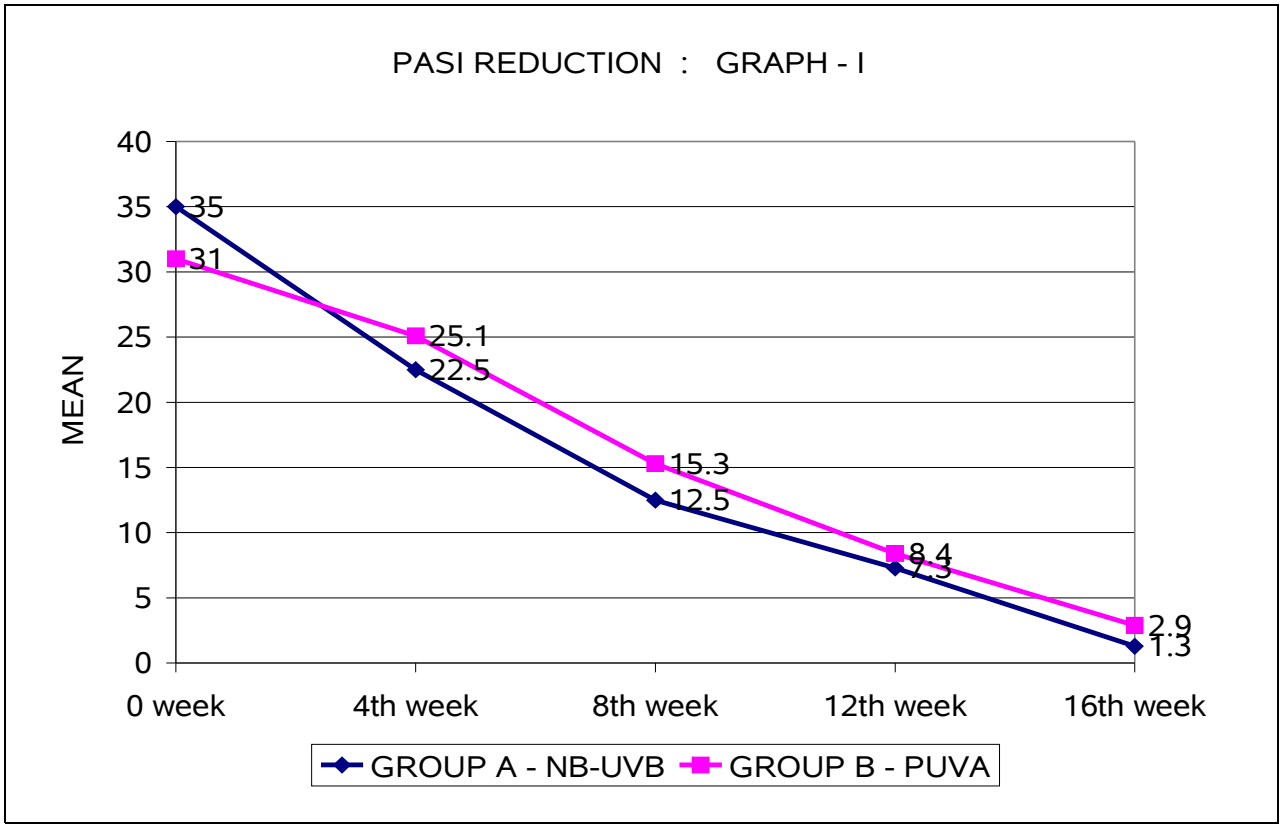


7) PASI Reduction

Group A

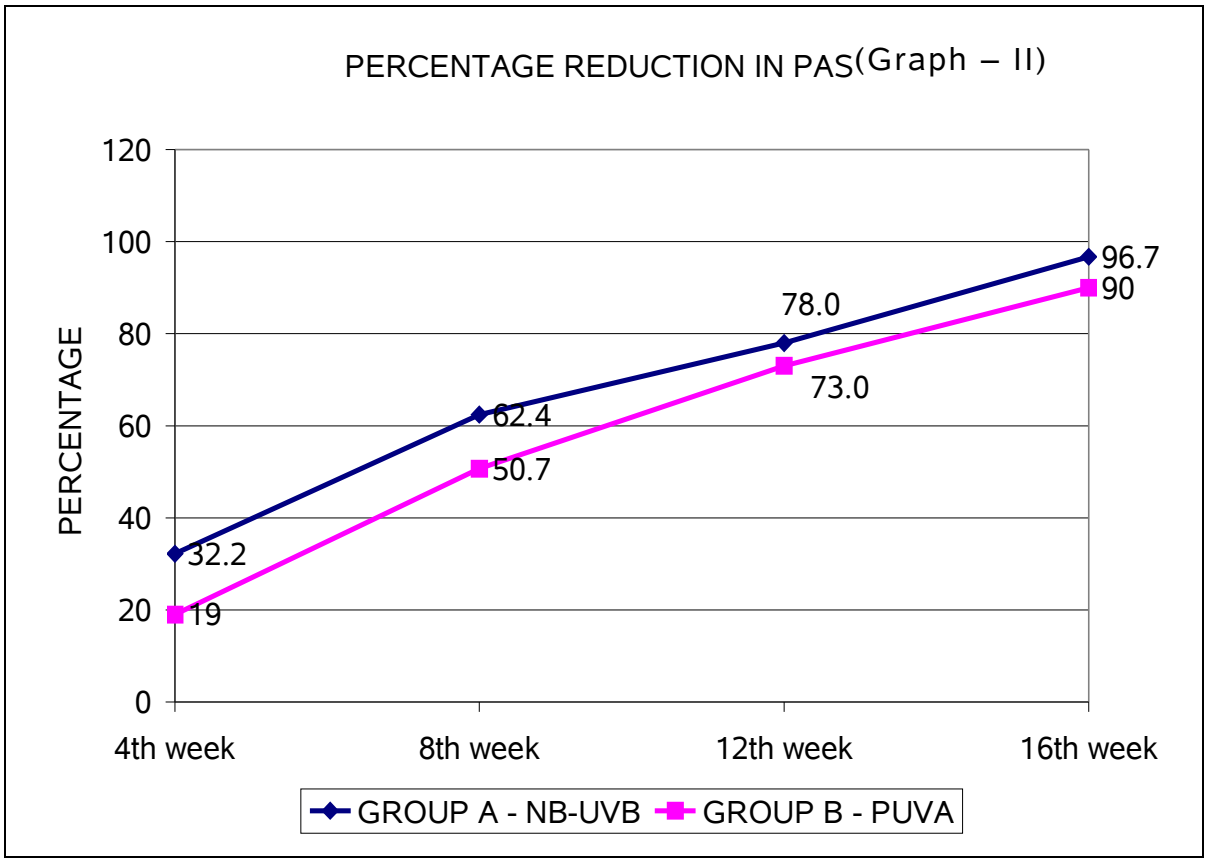
Mean PASI score at 0,4, 8,12 and16 weeks have been tabulated and its reduction is depicted in (Graph No.1)

No. of weeks	Mean PASI Score	
	Group A (NB-UVB)	Group B (PUVA)
0 week	35.0	31.0
4th week	22.5	25.1
8th week	12.5	15.3
12th week	7.3	8.4
16th week	1.3	2.9



8) Percentage reduction in PASI :

WEEKS	GROUP A (NB – UVB)	GROUP B (PUVA)
4th week	32.2%	19%
8th week	62.4%	50.7%
12th week	78%	73%
16th week	96.7%	90%



Graph II. depicts the percentage reduction in PASI scores in both groups at 4,8,12 and 16 weeks.

- From the table of PASI reduction we can see that the mean base line PASI scores in both group are 35.0 in group A and 31.0 in group B respectively.
- From the graph I – group A shows gradual reduction in PASI with a mean PASI score of 22.5 at 4 weeks, 12.5 at 8 weeks, 7.3 at 12 weeks and 1.3 at 16 weeks. These values corresponds to the percentage reduction of 32.2%, 62.4%, 78%, 96.7% at the end of 4,8,12 and 16 weeks respectively which is shown in graph II.
- In group B, the PASI score of 25.1, 15.3, 8.4, and 2.9 at the end of 4,8,12 and 16 weeks which corresponds to the percentage reduction of 19%, 50.7%, 73% and 90.0% at the end of 4,8,12 and 16 weeks respectively.

9) Mean cumulative UVB Dose and UVA Dose.

	GP – A (NB – UVB)	**GP – B PUVA**
Average No. of Exposure	36.06	30.9
Duration of treatment (weeks)	11.8	14.9
Mean cumulative Dose (J / cm^2)	27.2	223.3

The mean cumulative dose, average no. of exposures and total duration of treatment of UVB, & UVA has been tabulated.

10) Response to Therapy :

Based on the percentage of PASI the results were graded as good (75- 100%), moderate (50-75%) and poor (<50%).

Group A (NB – UVB):

Results	No. of Patients	Percentage	% reduction in PASI score at 16 weeks
Good	14	74 %	100 %
Moderate	2	11 %	>85 %
Poor response	3	16 %	30-40 %

Group B (PUVA):

Results	**No. of Patients**	**Percentage**	**% reduction in PASI score at 16 weeks**
Good	11	61 %	>96 %
Moderate	5	28 %	>72 %
Poor	2	11 %	10-30 %

In group A -

Out of 20 patients,

- 14 patients had good response.
- 2 patients moderately responded and
- 3 patients were resistant to NB- UVB therapy
- 1 patient discontinued therapy.

In group B –

Out of the 20 patients,

- 11 patients responded well
- 5 patients responded moderately and
- 2 patients were resistant to therapy, and
- 2 patients discontinued therapy.

12) Adverse effects :

Adverse effect	Group A	Group B
Pruritus	1	1
Erythema	1	1
Pigmentation	3	2
Gen weakness	1	0
Initial exacerbation	2	1

Out of the 40 patients, the following adverse effects were noted.

- Exacerbation pruritus in 2 patients,
- Erythema in 2 patients
- Generalised pigmentation in 5 patients
- Generalised weakness in 1 patient
- Initial exacerbation in 3 patients, which gradually resolved after continuation of the therapy.

DISCUSSION

Since the introduction of Narrow Band-UVB to the field of phototherapy for Psoriasis, many studies reported that it is more effective and safer than PUVA and has a good effect on quality of life.

However others recommended the superiority of PUVA. On the other side some reports revealed that both therapies have equal effect.

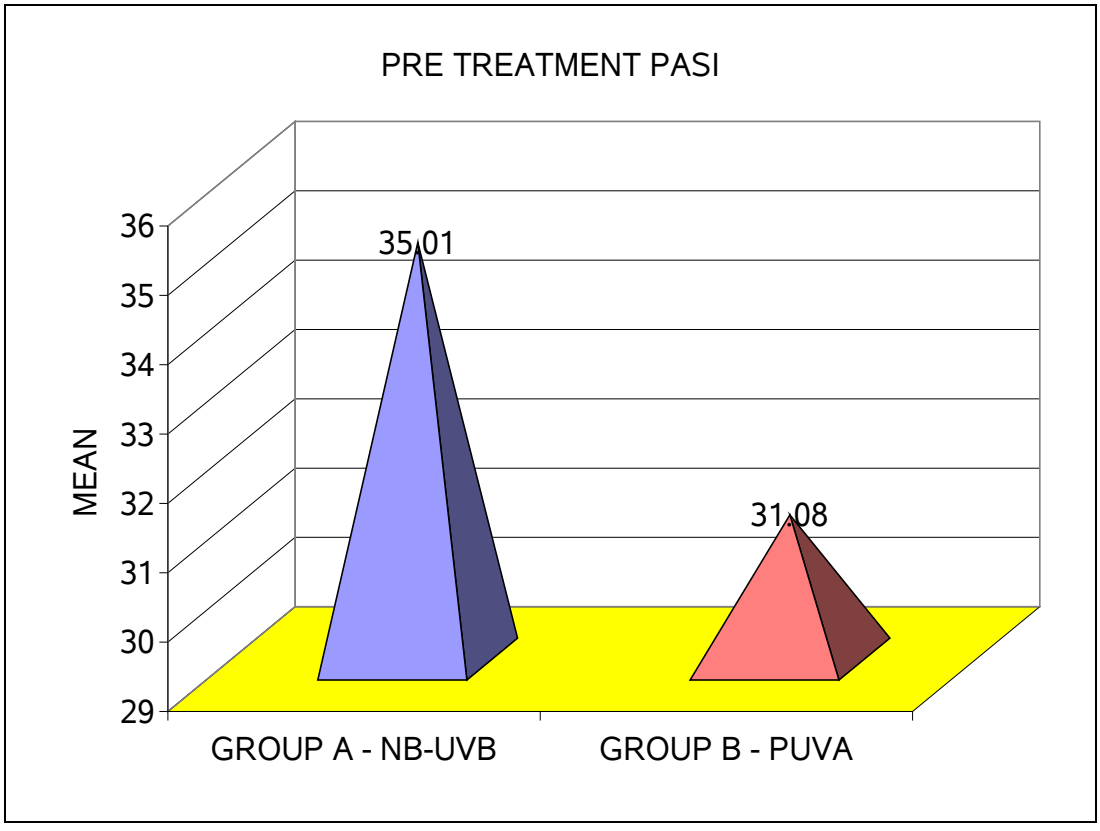
To gain our own experience, this study was designed to compare NB-UVB therapy to PUVA therapy in the management of chronic plaque type psoriasis.

TREATMENT RESPONSE

We found that NB-UVB therapy is slightly superior to PUVA therapy. The final evaluation involved comparison of both treatments according to the response, cumulative dose and adverse effects. The response of patients ranged from excellent (75 % or More decrease in PASI score) to no response (Fixed or only Slight decrease in PASI score). Patients showed slightly increased response to NB-UVB compared to PUVA. These results are consistent with some of the studies.[62,63,64]

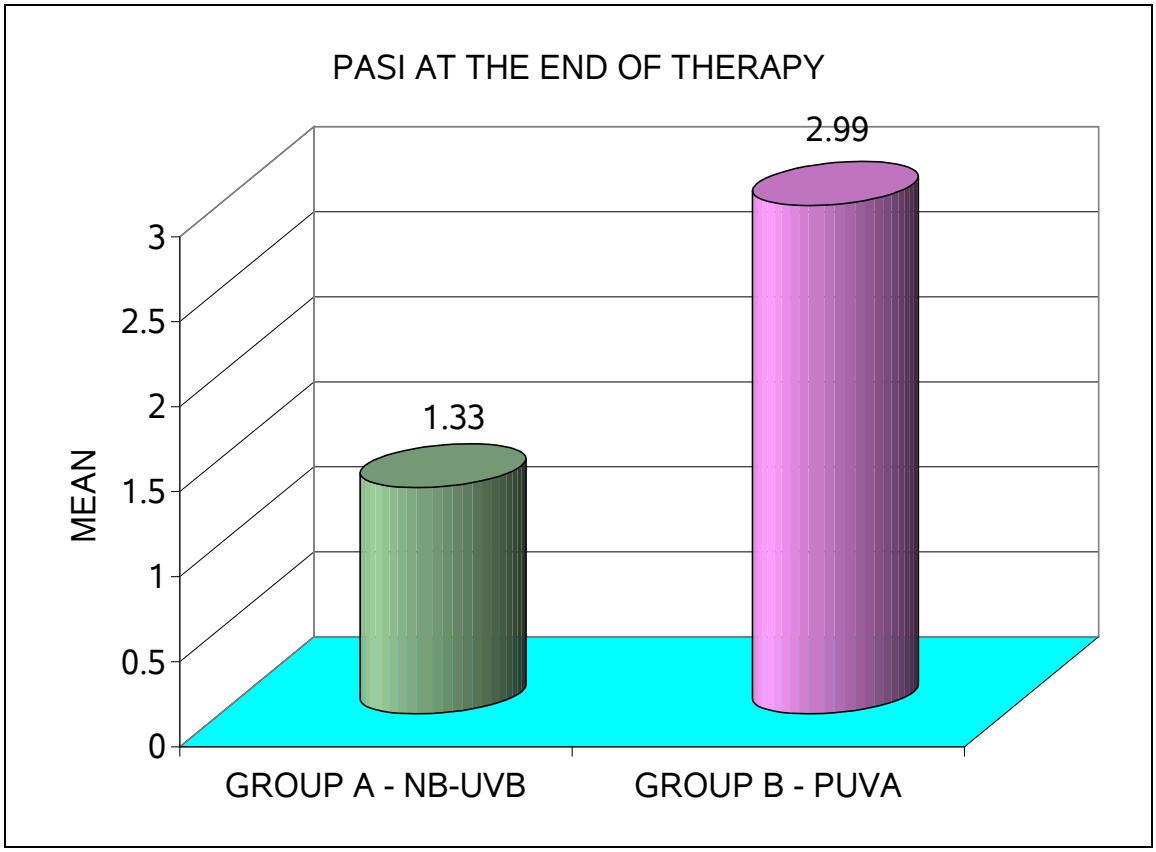
PRETREATMENT PASI

Group	Range	Mean	SD	P
A (NB-UVB)	21.9 to 59.9	35.01	8.78	0.185 (Not Significant)
B (PUVA)	17 – 52.2	31.08	9.58	



PASI AT THE END OF THERAPY

Group	Range	Mean	SD	P
A	0 – 10.5	1.3	3.57	0.0436
B	0 – 11.8	2.99	4.2	**(Significant)**



SIDE EFFECTS

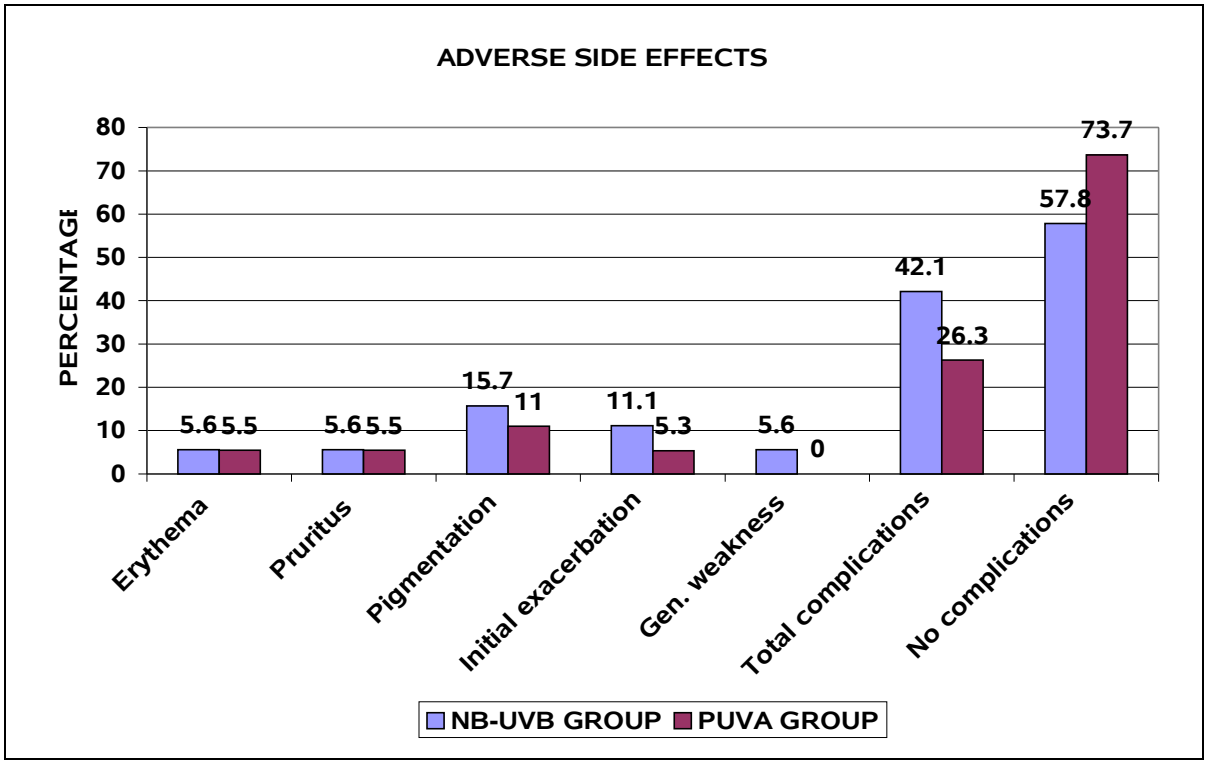
In our study, side effect like generalised pigmentation, erythema, pruritus, generalised weakness and initial exacerbation were noted. No significant difference in the side effect between the two therapies.

In our study, significant erythema is noted only in two patients, one patient in NB-UVB and other in PUVA but in many other studies, common side effect of UV therapy is erythema. This significant difference is probably because, all of our patients are of skin type IV and V.

Exacerbated pruritus is noted in two of our patients initially, which subsided after continuation of therapy. It is assumed to be related to prostaglandin relies.

Generalised pigmentation is noted in three of our patients on NB-UVB and two patients on PUVA. It shows an higher incidence than in other studies, pigmentation is a common complication in dark skinned individuals.[65] This may be a reason for increase incidence of pigmentation in our patients.

Adverse side effects	NB-UVB Group		PUVA Group	
	No.	%	No.	%
Erythema	1	5.6	1	5.5
Pruritus	1	5.6	1	5.5
Pigmentation	3	15.7	2	11
Initial exacerbation	2	11.1	1	5.3
Gen. weakness	1	5.6	0	0
Total complications	8	42.1	5	26.3
No complications	11	57.8	14	73.7
'p'	**0.3787 Not significant**			



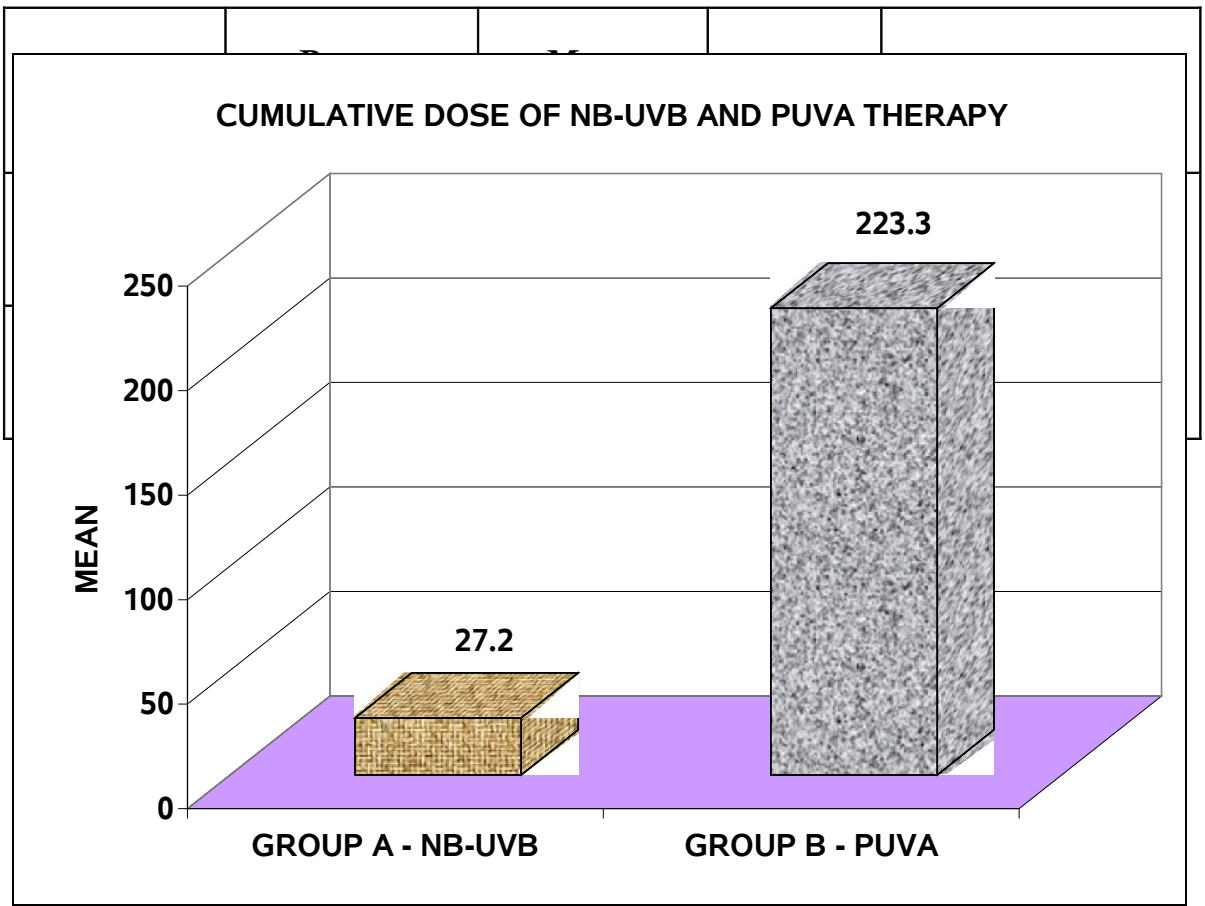
TREATMENT RESISTANCE

Treatment resistance is noted in three of our patients in NB-UVB and two patients in PUVA. It correlates with the long duration of illness and it has no correlation with PASI score at the initiation of therapy.

TOTAL CUMULATIVE DOSE

Mean cumulative dose of NB-UVB in our study was of 27.2 J/cm^2 and in PUVA it was 223.3 J/cm^2. Cumulative dose is significantly higher in patients in PUVA therapy when compared to NB-UVB, Which may explain the carcinogenic potential of PUVA when compared to NB-UVB.

CUMULATIVE DOSE OF NB-UVB AND PUVA THERAPY



CONCLUSION

NB-UVB phototherapy is superior when compared to PUVA in the treatment of plaque type psoriasis.

- ✓ The response to therapy is better with NB-UVB than with PUVA therapy
- ✓ The therapeutic effect of NB-UVB is achieved with significantly lower cumulative dose which means a lower risk for incidence of long term complication, with special reference to skin malignancy.
- ✓ NB-UVB is more convenient for the patient and is less time consuming.
- ✓ No exogenous photosensitizer is used NB-UVB therapy, thus prevents the side effect of psoralen.
- ✓ No need for eye and skin protection after the session in NB-UVB phototherapy.
- ✓ It can be used in pregnancy, lactation and it is useful and well tolerated treatment for children, but concerns remain regarding its long term side effects.

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PROFORMA

Name :

Age / Sex :

Case No :

Psoriasis Clinic No :

Marital Status :

Occupation :

Address :

HISTORY

Duration : Months/ Yrs

Itching : Yes/No

Previous Treatment : Topical/ Systemic

- EXACERBATION WITH :

  Cold Climate

  Sunlight

  Infection

  Trauma

  Drugs

  Emotional Factors

  Puberty

  Pregnancy

  Menopause

- Preceeding sore throat - Yes/No
- Alcohol Intake - Yes/No
- Smoking - Yes/No
- Systemic illness like Diabetes, Hypertension, Tuberculosis or Renal Diseases
- Pregnancy - Yes/No
- Lactation - Yes/No

- Past history of Photosensitivity, Cutaneous Malignancies, or Radiotherapy
- Family history of Psoriasis

DRUG INTAKE

NAME OF THE DRUG	YES	NO	DURATION OF TREATMENT	
			YEAR	MONTH
Lithium				
β blockers				
Antimalarials				
Steroid Withdrawal				
Clonidine				
Potassium iodine				
Amiodarone				
Digoxin				
Trazadone				
Gemfibrozil				
Penicillin				
Terfenadine				
NSAIDs				
Natural remedies.				

EXAMINATION

General

Systemic

CVS
RS
Abdomen
CNS

Dermatological :

Morphology of the lesions
Sites of involvement

Auspitz Sign **:** No/Yes

Clinical Type : - Chronic Stable Plaque
- Scalp
- Palmoplantar
- Others-Guttate / Erythrodermic /
- Unstable / Flexural/ Sebo/Rupioid

Nail Changes : - None
- Pitting
- Oycholysis
- Subungual hyperkeratosis
- Oil Drop Sign
- Splinter Hemorrhages
- Ridges
- Yellow discoloration

Joint Involvement : - None
- Classical DIP joint arthritis
- Asymmetric Oligoarthritis
- Symmetric Polyarthritis
- Axial arthritis
- Arthritis Mutilans

Focal Sepsis : ENT/ Dental/ Others

Area and severity assessment by PASI scoring:

Erythema/Infiltration/Desquamation Scoring			**Area Scoring**
0	-	Nil	0 – Nil
1	-	Mild	1 – 0%-9%
2	-	Moderate	2 – 10 % - 29%
3	-	Severe	3 – 30 % - 49 %
4	-	Very Severe	4 – 50 % - 69 %
			5 – 70 % - 89 %
			6 – 90 % - 100 %

$$PASI = 0.1 (E_H + I_H + D_H) A_H + 0.2 (E_U + I_U + D_U) A_U$$
$$0.3 (E_T + I_T + D_T) A_T + 0.4 (E_L + I_L + D_L) A_L$$

INVESTIGATION

Blood Sugar : Urea : Creatinine :

Blood VDRL: HIV:

Ophthalmic Examination

Date							
LFT							
SGOT							
SGPT							
SAP							
Total Proteins							
Total Bilirubin							
Haemogram							
TC							
DC							
Hb							
ESR							

UV CHART

Date	Cycle	Dose	Duration	Cumulative Dose	Side Effects

KEY TO MASTER CHART

P	-	Pitting
O	-	Onycholysis
SUH	-	Subugual Hyperkeratosis
YD	-	Yellow Discolouration
SE	-	Side effects
Pi	-	Generalised pigmentation
Itc	-	Itching
Ery	-	Erythema
Gw	-	Generalised weakness
Ie	-	Initial Exacerbation

BEFORE TREATMENT

AFTER NB-UVB THERAPY

EXACERBATION AND ERYTHEMA INDUCED BY

PUVA THERAPY

CO UVA)

BEFORE TREATMENT

AFTER NB-UVB THERAPY

BEFORE TREATMENT

AFTER PUVA THERAPY

BEFORE TREATMENT – PLAQUE TYPE PSORIASIS

IN A 12 YEAR OLD GIRL

AFTER NB-UVB THERAPY